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Intrafamilial Spread of *Helicobacter Pylori* Infection

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Abstract

Helicobacter pylori (*H. pylori*) is the main cause of upper gastrointestinal diseases including gastric cancer. Humans are the principal reservoir of the infection. Intrafamilial spread of *H. pylori* is poorly documented. We compared the prevalence of infection within household contacts of *H. pylori*-infected patients with that of subjects with non-alcoholic fatty liver disease referred to our GI unit from January to October 2015. We studied 95 household contacts (49 M and 46 F, median age 38 years, age range 17-72 years) of 40 dyspeptic patients with *H. pylori* infection based on both rapid urease test and histology. As a control group, we studied 95 subjects (42 M and 53 F, median age 37 years, range 16-73 years) referred to our outpatient clinic for non-alcoholic fatty liver disease. We found *H. pylori* infection in 41/95 household contacts (43.2%), whereas only 27/95 (28.4%) control subjects resulted infected. This difference was statistically significant (p= 0.034). Based on the results of this study, subjects with *H. pylori* infection represent an important source of infection within their families. We suggest testing for *H. pylori* all household contacts of *H. pylori*-infected patients to prevent spreading of infection, especially in areas where the prevalence of *H. pylori*-related upper gastrointestinal malignancies is high.

Keywords: Gastric cancer; *Helicobacter pylori*

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Introduction

H. pylori are associated with chronic gastritis, peptic ulcer, gastric cancer, and gastric lymphoma [1]. Prevalence of *H. pylori* infection is higher in developing countries when compared to developed countries, where prevalence among adults is around 80-90% and <40%, respectively. Socio-economic factors such as low income, high household density of children and use of stove for heating were found to be important risk factors for *H. pylori* infection among preschool and school children [2,3]. Family setting could be a transmission place very important; a study of Brazil pointed out *H. pylori* positive mothers are a strong and independent risk factor for *H. pylori* infection of their children [4]. Intrafamilial spread of infection is little studied [5-7]. A study of 39 families using molecular typing of strains found the same strains in a large proportion of sibs (29 of 35, 81%), mother-offspring strain concordance was observed in 10 of 18 (56%) of the families, whereas father-offspring strain concordance was not occurring. Clustering of siblings strains was also observed in six families where the mother harboured her own strain. Spouses were infected with the same strain in 5 of 23 (22%) of cases, pointing also to a possible relevance of spouse-to-spouse transmission [8]. We wanted, therefore, evaluate there was a real increase in the prevalence of family members of patients with *H. pylori* infection compared to a control group.

Materials and Methods

Population

The study population consisted of 95 household contacts of 40 consecutive outpatients dyspeptic patients with *H. pylori* infection, based on both rapid urease test and histology [9], referred to our GI unit from January to October 2015. The control group consisted of 95 consecutive subjects referred to our outpatient clinic for non-alcoholic fatty liver disease in the same period of time. Exclusion criteria were age <18 years, pregnancy, use of proton pump inhibitors (PPIs), histamine H_2 receptor antagonists, antibiotics or non-steroidal anti-inflammatory drugs (NSAIDs) in the last two months.

Statistical analysis

Statistical analyses were performed using Statistical Program for Social Sciences (SPSS®) ver.16.0 for Macintosh® (SPSS Inc., Chicago, Illinois, USA). Student t-test and Mann-Whitney U test were performed in order to compare continuous variables, chi-square with Yates correction, and Fisher-exact test to compare categorical variables. Statistical significance was defined as p< 0.05 in a two-

Table 1: Distribution of *H. pylori* infection according to degree of relationship.

	UBT	
	Positive	Negative
Spouse	17/34	17/34
Offspring	14/46	32/46
Parents	2/4	2/4
Siblings	5/6	1/6
Nephew	3/3	0/3
In laws	0/2	2/2

tailed test with a 95% Confidence Interval.

Evaluation of *H. pylori* infection

H. pylori status in the 95 household contacts and in the 95 control subjects was assessed by ^{13}C-Urea Breath Test (UBT) and determination of *H. pylori* stool antigen (HpSA). Subjects were considered to be infected if both tests were positive. Tests were performed after at least two months without antibiotics, PPIs, histamine H2 receptor or NSAIDs therapy. Smoking was not allowed for 12 h before the ^{13}C-UBT. The ^{13}C-UBT was performed after an overnight fast. A baseline breath sample was obtained, and 100 mg of 13C urea with citric acid (1.4 g) was administered as an aqueous solution (Expirobacter, SOFAR, Milan, Italy). Another breath sample was collected 30 min later. The test was considered positive if the difference between the baseline sample and the 30-min sample exceeded 5.0 parts/1000 of $^{13}CO_2$. All breath tests were analyzed at the same laboratory by using a single gas isotope ratio mass spectrometer (ABCA, Europe Scientific, Crewe, UK). HpSA is an enzyme immunoassay test for in vitro qualitative detection of *H. pylori* antigens in human stool (Premier Platinum HpSA, manufactured by Meridian Diagnostics Inc., USA), and it was performed at the same day on which the patient performed ^{13}C-UBT [10].

This work was carried out in accordance to ethical guidelines of 1964 Declaration of Helsinki and all patients gave informed written consent prior to their inclusion in the study.

Results and Discussion

Ninety household contacts were 49 M and 46 F, median age 38 years, age range 17-72 years. Of the 95 household contacts, thirteen subjects had upper gastrointestinal (GI) symptoms (4 dismotility-like dyspepsia, 5 ulcer-like dyspepsia, 4 heartburn and regurgitation) and 12 had irritable bowel syndrome. The remaining had no upper or lower GI symptoms. 95 control subjects were 42 M and 53 F, median age 37 years, range 16-73 years. Of the 95 control subjects, 19 had upper gastrointestinal symptoms (10 dismotility-like dyspepsia, 6 ulcer-like dyspepsia, 3 heart burn and regurgitation) and 10 irritable bowel syndromes. The remaining did not complain of any upper or lower GI symptoms. These two groups were homogeneous for sex and age and for socio-economic status as assessed by level of education and household size. We found *H. pylori* infection in 41/95 household contacts (43.2%), whereas only 27/95 (28.4%) control subjects resulted infected. This difference was statistically significant (p= 0.034). In seven families, we did not detect any *H. pylori* infection while in 17 families all of the family members were *H. pylori*-infected. The distribution of *H. pylori* infection according to degree of relationship is indicated in the Table 1. Based on the results of this study, subjects with *H. pylori* infection represent an important source of infection within their families. We suggest to test for *H. pylori* all household contacts of *H. pylori*-infected patients to prevent spreading of infection, especially in areas where the prevalence of *H. pylori*-related upper gastrointestinal malignancies is high [11,12].

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